

ETEC colonization factors modulate the antigen presentation function of porcine intestinal mononuclear phagocytes

Bert DEVRIENDT, Kim BAERT, Yu LUO, Bruno M. GODDEERIS, Eric COX

Laboratory of Immunology, Faculty of Veterinary Medicine, University of Ghent, 9820 Merelbeke, Belgium

Enterotoxigenic *E. coli* (ETEC) are still a major cause of traveller's diarrhea and intestinal infections in children in less affluent countries. In addition, ETEC infections lead to postweaning diarrhea in piglets and result in significant economic losses in swine production. This intestinal pathogen displays colonization factors or fimbriae on its surface enabling the colonization of the small intestinal epithelia. In swine, F4 and F18 fimbriae are frequently associated with ETEC-induced diarrhea. As opposed to F4 fimbriae, oral immunisation with F18 fimbriae fails to protect animals from a challenge infection. Besides structural differences and a different uptake mechanism by the intestinal epithelium of F18 (M-cell dependent transport) and F4 fimbriae (M-cell and enterocyt-dependent transport), these fimbriae could differently modulate the function of intestinal phagocytes. Indeed, F18 fimbriae drastically diminished the antigen presentation capacity of mononuclear phagocytes (MHCII⁺SIRP α ^{hi}) isolated from the small intestinal lamina propria. As F18 fimbriae bind glycosphingolipids and presumably disrupt lipid raft formation, the impaired antigen presentation ability of the intestinal phagocytes could result from a F18 fimbriae-mediated deformation of the immunological synapse. These results could accelerate the development of an improved F18⁺ ETEC vaccine and hint at novel immune evasive mechanisms employed by bacterial pathogens.